Pediatric Headache

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Objectives

Epidemiology

Classification

Migraine

Tension type headache

Clinical approach to a child with headache

Epidemiology

Headache is common in children and occurs at least annually in approximately 40% of children by age of 7 years of age and 50% of children by 15 years.

The most frequent type of recurrent headache in childhood is migraine; in adolescents, tension headaches are the most common cause of frequent headache.

Race- and sex-related demographics

No specific report exists regarding differential incidence of headache by race in children, but migraine frequency in adults in the US declines from whites to African-Americans to Asians.

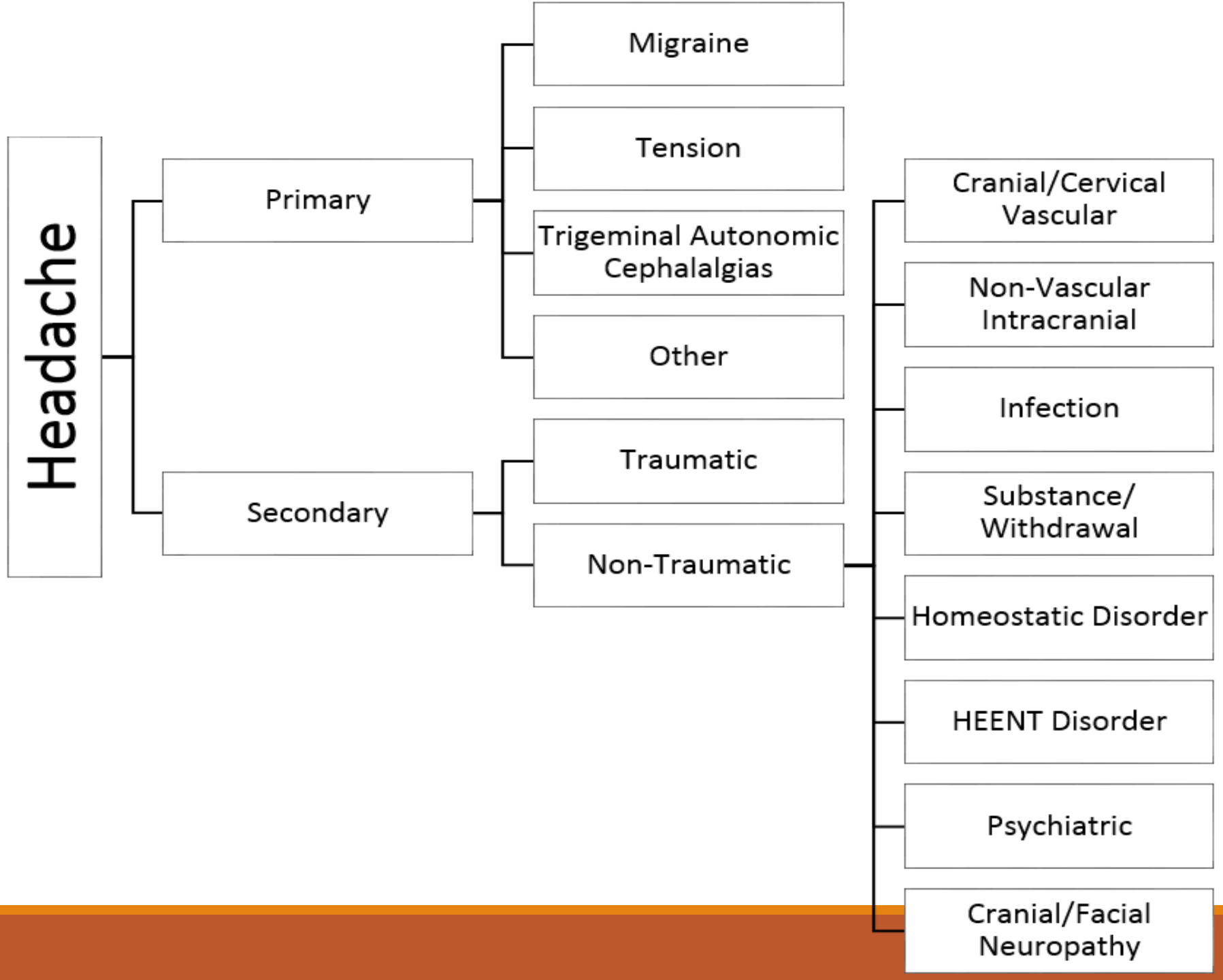
Approximately 60% of all children with migraines before puberty are male. Thereafter, the relationship is inversed, with 3 times more female than male migraineurs in adulthood.

Etiology

Because the brain is insensate, headache is due to the stimulation of pain-sensitive nerve fibers in large cerebral arteries and veins, the periosteum of the skull, the muscle and skin of the scalp, the sinus mucosa, the temporomandibular joint, the teeth, or the gingiva.

Classification of Headache, ICHD-3

table


Headache
Primary
Secondary
Migraine
Tension
Trigeminal Autonomic Cephalalgias
Other
Traumatic
Non-Traumatic
Cranial/Cervical Vascular
Non-Vascular Intracranial
Infection
Substance/ Withdrawal
Homeostatic Disorder
HEENT Disorder
Psychiatric
Cranial/Facial Neuropathy

Migraine

Epidemiology

Migraine is the most frequent type of recurrent headache It is reported by up to 10.6% of children between the ages of 5 and 15 years and up to 28% of adolescents

They convert to chronic daily headaches in 1% of children

The risk of conversion is higher when the headaches occur more than 15 days a month

Migraines have a large impact on the child's development, school performance as well as psychosocial aspects of their life.

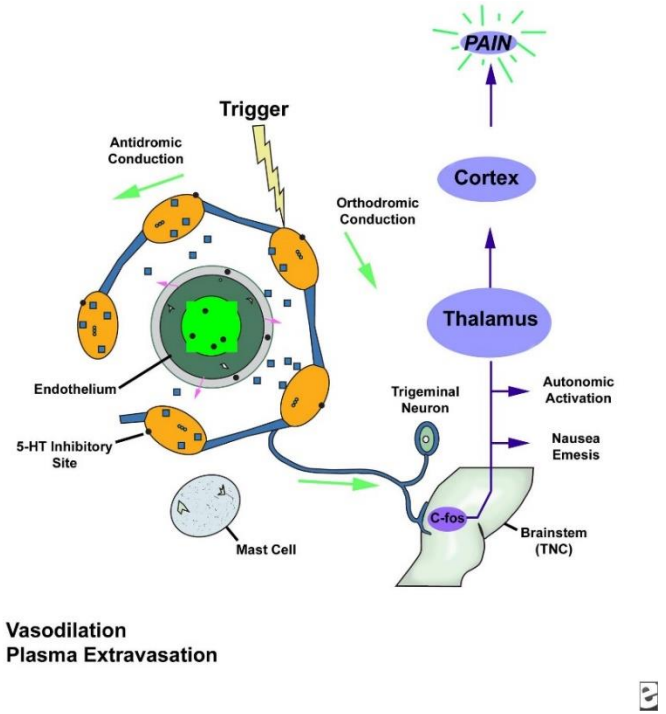
Migraine pathophysiology:

Although much remains to be discovered, the pain in migraine attacks is multifactorial.

- Trigeminovascular system activation
- Cortical spreading depression
- Genetic predisposition

Trigeminovascular system activation

Trigeminovascular system. The trigeminal nerve fibers around basal cerebral and meningeal vessels are triggered (various stimuli are possible), and a vicious cycle starts in which the nerve terminals release calcitonin gene-related peptide (CGRP), substance P, vasoinhibitory peptide (VIP), and other mediators of local neurogenic inflammation and vasodilatation. The latter further stimulates the nerve endings. On the other end of the nerve, painful messages are transmitted toward central centers, including thalamus and cortex, and the sensation of pain arises



Cortical spreading depression

Another mechanism thought to result in migraine headache has its origin in the brain stem.

The onset of the aura in migraine headache is thought to be mediated by cortical spreading depression (CSD)—caused by neuronal activation followed by suppression—which spreads over the cortical surface.

A simultaneous change occurs in cerebral blood flow, characterized by hyperperfusion, followed by hypoperfusion.

Genetic predisposition

About 70-90% of pediatric patients with migraine have a family history of migraine headache.

Some individuals with familial hemiplegic migraine (FHM) have been found to have several genetic mutations in ion channels responsible for neurotransmitter release within the CNS, which may ultimately affect cortical excitability.

Migraine Headache

In children, the headache is more often bifrontal, bitemporal, or generalized than unilateral.

Onset is gradual, occurring as a cascade of events over hours or days

Migraine headache is throbbing or pulsatile in nature, especially as it increases in intensity.

The migraine attack is often accompanied by nausea photophobia and phonophobia. Many children have vomiting that occurs once or repetitively, autonomic symptoms may also occur with the headache.

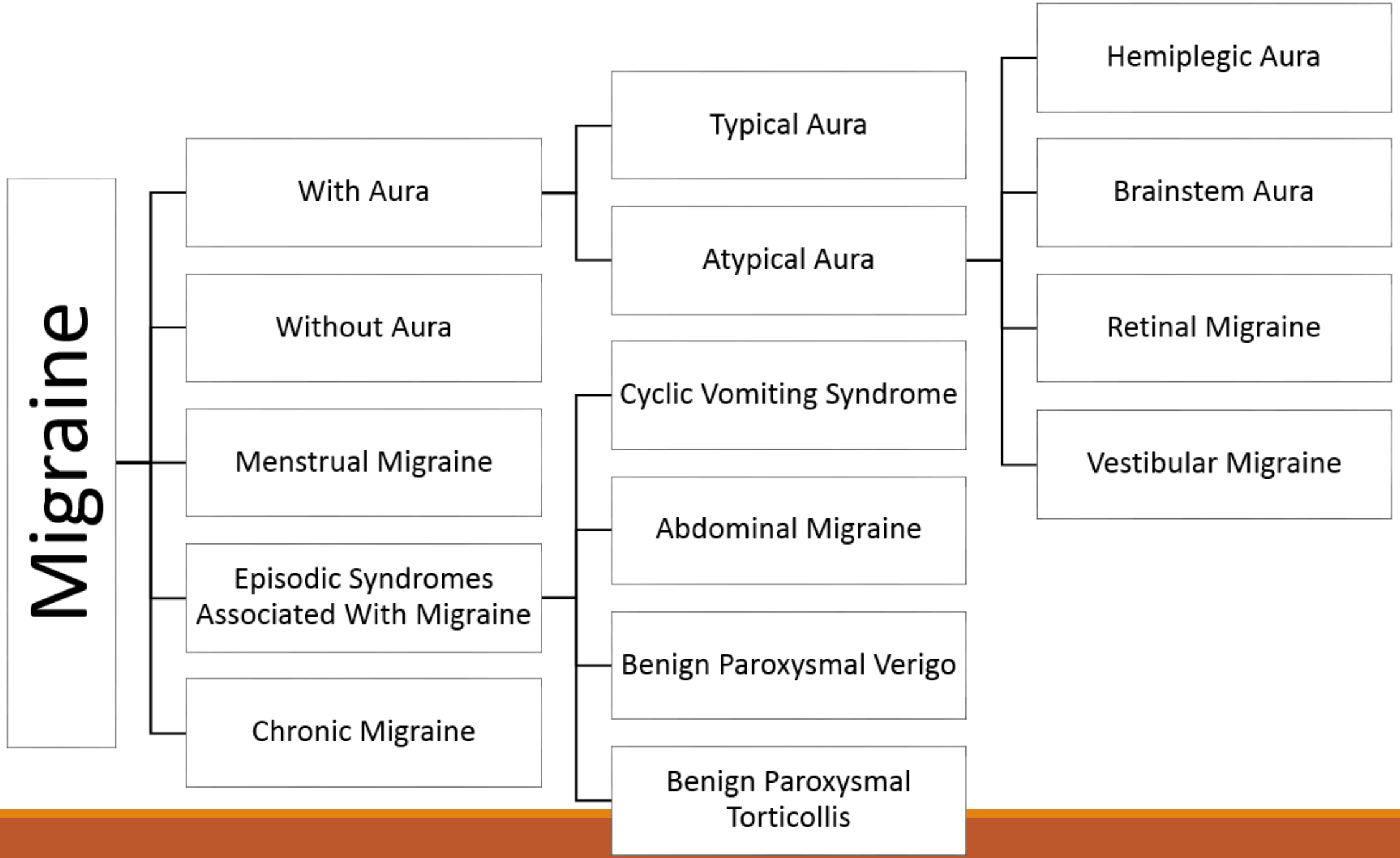
Patients typically want to lie down in a dark, quiet room and may obtain relief with sleep.

In toddlers, symptoms appreciated by caregivers include pallor, decreased activity, and vomiting.

Migraine Headache cont.

The duration of migraine headache tends to be shorter in younger compared with older children or adults.

Migraine episodes may be triggered by variety of factors eg: stress, lack of sleep, excitement, menstruation, food triggers are much less common than in adults.


Migraine
With Aura
Without Aura
Menstrual Migraine
Episodic Syndromes Associated With Migraine
Chronic Migraine
Typical Aura
Atypical Aura
Cyclic Vomiting Syndrome
Abdominal Migraine
Benign Paroxysmal Verigo
Benign Paroxysmal Torticollis
Hemiplegic Aura
Brainstem Aura
Retinal Migraine
Vestibular Migraine

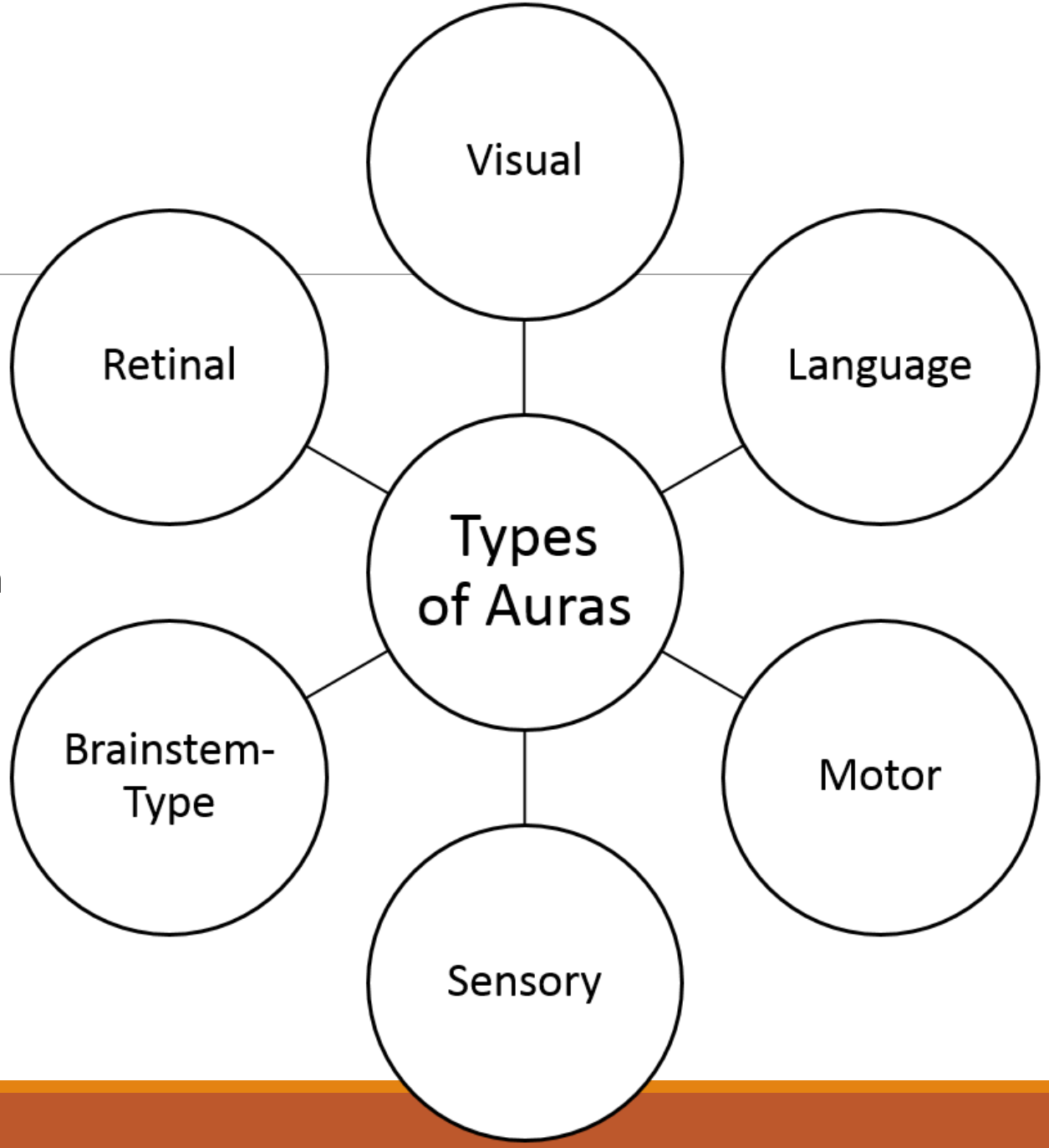
Migraine Aura

Aura, when it occurs, is most often visual.

The most typical duration of aura is 5 to 60 minutes.

The importance of the aura lasting longer than 5 min is to differentiate the migraine aura from a seizure with a postictal headache

The 60 min maximal duration is to separate migraine aura from the possibility of a more prolonged neurologic event such as a transient ischemic attack.



Atypical Auras

Include:

-Hemiplegic Aura

-Basilar Aura (Vertigo or lower cranial nerve symptoms)

-Distortion (Alice in Wonderland Syndrome)

These are very rare forms of auras and their presence warrants further investigation.

Hemiplegic migraine

Transient unilateral weakness, usually lasts only a few hours but may persists for days

Headache and may be other typical aura

Might be associated with encephalopathy, ataxia, seizure

Familial hemiplegic migraine is an autosomal dominant disorder with mutation described in 4 separate genes:

(1) CACNA1A, (2) ATP1A2, (3) SCNA1,(4) PRRT2

Sporadic form is described

Table 595-2 Migraine Without Aura

A. At least 5 attacks fulfilling criteria B to D
B. Headache attacks lasting 4-72 hr (untreated or unsuccessfully treated)
C. Headache has at least 2 of the following 4 characteristics:
    1. Unilateral location
    2. Pulsating quality
    3. Moderate or severe pain intensity
    4. Aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D. During headache at least 1 of the following:
    1. Nausea and/or vomiting
    2. Photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis

Table 595-3 Migraine with Typical Aura

A. At least 2 attacks fulfilling criteria B and C
B. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but no motor, brainstem or retinal symptoms
C. At least 2 of the following 4 characteristics:
    1. At least 1 aura symptom spreads gradually over 5 or more minutes, and/or 2 or more symptoms occur in succession
    2. Each individual aura symptom lasts 5-60 minutes
    3. At least 1 aura symptom is unilateral
    4. The aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded

Table 595-4 Migraine with Brainstem Aura

A. At least 2 attacks fulfilling criteria B to D
B. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but no motor or retinal symptoms
C. At least 2 of the following brainstem symptoms:
    1. Dysarthria
    2. Vertigo
    3. Tinnitus
    4. Hypacusis
    5. Diplopia
    6. Ataxia
    7. Decreased level of consciousness
D. At least 2 of the following 4 characteristics:
    1. At least 1 aura symptom spreads gradually over 5 or more minutes, and/or 2 or more symptoms occur in succession
    2. Each individual aura symptom lasts 5-60 minutes
    3. At least 1 aura symptom is unilateral
    4. The aura is accompanied, or followed within 60 minutes, by headache
E. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

Table 595-5 Vestibular Migraine with Vertigo

A. At least 5 episodes fulfilling criteria C and D
B. A current or past history of 1.1 *Migraine without aura* or 1.2 *Migraine with aura*
C. Vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 hr
D. At least 50% of episodes are associated with at least 1 of the following 3 migrainous features:
    1. Headache with at least 2 of the following 4 characteristics:
        a. Unilateral location
        b. Pulsating quality
        c. Moderate or severe intensity
        d. Aggravation by routine physical activity
    2. Photophobia and phonophobia
    3. Visual aura
E. Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder

Table 595-6 | Chronic Migraine

A. Headache (tension-type-like and/or migraine-like) on 15 or more days per month for more than 3 mo and fulfilling criteria B and C

B. Occurring in a patient who has had at least 5 attacks fulfilling criteria B to D for 1.1 *Migraine without aura* and/or criteria B and C for 1.2 *Migraine with aura*

C. On 8 or more days per month for more than 3 mo, fulfilling any of the following:
   1. Criteria C and D for 1.1 *Migraine without aura*
   2. Criteria B and C for 1.2 *Migraine with aura*
   3. Believed by the patient to be migraine at onset and relieved by a triptan or ergot derivative

D. Not better accounted for by another ICHD-3 diagnosis

Migraine Treatment

The American Academy of Neurology established useful practice guidelines for the management of migraine as follows:

1. **Reduction of headache frequency, severity, duration, and disability**
2. **Reduction of reliance on poorly tolerated, ineffective, or unwanted acute pharmacotherapies**
3. **Improvement in quality of life**
4. **Avoidance of acute headache medication escalation**
5. **Education and enabling of patients to manage their disease to enhance personal control of their migraine**
6. **Reduction of headache-related distress and psychologic symptoms.**

Treatment :

ACUTE MIGRAINE:

Management of an acute attack is to provide headache freedom as

quickly as possible with return to normal function.

This mainly includes 2 groups of medicines:

1-nonsteroidal anti-inflammatory drugs (NSAIDs) . Or paracetamol

2- triptans.

Most migraine headaches in children will respond to appropriate doses of NSAIDs when administered at the onset of the headache attack.

DRUG	DOSE	MECHANISM	SIDE EFFECTS	COMMENTS
ACUTE MIGRAINE				
Analgesics				
Acetaminophen	15 mg/kg/dose	Analgesic effects	Overdose, fatal hepatic necrosis	Effectiveness limited in migraine
Ibuprofen	7.5-10 mg/kg/dose	Antiinflammatory and analgesic	GI bleeding stomach upset, kidney injury	Avoid overuse (2-3 times per wk)
Triptans				
Almotriptan* (ages 12-17 yr)	12.5 mg	5-$HT_{1b/1d}$ agonist	Vascular constriction, serotonin symptoms such as flushing, paresthesias, somnolence, GI discomfort	Avoid overuse (more than 4-6 times per mo)
Eletriptan	40 mg	Same	Same	Avoid overuse (more than 4-6 times per mo)
Frovatriptan	2.5 mg	Same	Same	May be effective for menstrual migraine prevention Avoid overuse (more than 4-6 times per mo)
Naratriptan	2.5 mg	Same	Same	May be effective for menstrual migraine prevention Avoid overuse (more than 4-6 times per mo)
Rizatriptan* (ages 6-17 yr)	5 mg for child weighing <40 kg, 10 mg	Same	Same	Available in tablets and melts Avoid overuse (more than 4-6 times per mo)
Sumatriptan	Oral: 25 mg, 50 mg, 100 mg Nasal: 10 mg SC: 6 mg	Same	Same	Avoid overuse (more than 4-6 times per mo)
Zolmitriptan	Oral: 2.5 mg, 5 mg Nasal: 5 mg	Same	Same	Available in tablets and melts Avoid overuse (more than 4-6 times per mo)

Prophylaxis :

Calcium Channel Blockers : Flunarizine.

Anticonvulsants :Valproic acid, Topiramate, Levetiracetam, Gabapentin.

Antidepressants : Amitriptyline.

Antihistamines : Cyproheptadine.

Antihypertensive : Propranolol

DRUG	DOSE	MECHANISM	SIDE EFFECTS	COMMENTS
PROPHYLAXIS (NONE APPROVED BY FDA FOR CHILDREN)				
Calcium Channel Blockers				
Flunarizine†	5 mg hs	Calcium channel blocking agent	Headache, lethargy, dizziness	May ↑ to 10 mg hs
Anticonvulsants				
Valproic acid	20 mg/kg/24 hr (begin 5 mg/kg/24 hr)	↑ Brain GABA	Nausea, pancreatitis, fatal hepatotoxicity	↑ 5 mg/kg every 2 wk
Topiramate	100-200 mg divided bid	↑ Activity of GABA	Fatigue, nervousness	Increase slowly over 12-16 wk
Levetiracetam	20-60 mg/kg divided bid	Unknown	Irritability, fatigue	Increase every 2 wk starting at 20 mg/kg divided bid
Gabapentin	900-1800 mg divided bid	Unknown	Somnolence, fatigue aggression, weight gain	Begin 300 mg, ↑ 300 mg/wk
Antidepressants				
Amitriptyline	1 mg/kg/day	↑ CNS serotonin and norepinephrine	Cardiac conduction, abnormalities and dry mouth, constipation, drowsiness, confusion	Increase by 0.25 mg/kg every 2 wk Morning sleepiness reduced by administration at dinnertime
Antihistamines				
Cyproheptadine	0.2-0.4 mg/kg divided bid; max: 0.5 mg/kg/24 hr	H_1-receptor and serotonin agonist	Drowsiness, thick bronchial secretions	Preferred in children who cannot swallow pills; not well tolerated in adolescents
Antihypertensive				
Propranolol	10-20 mg tid	Nonselective β-adrenergic blocking agent	Dizziness, lethargy	Begin 10 mg/24 hr ↑ 10 mg/wk (contraindicated in asthma and depression)
Others				

Sever intractable migraine

6-7% of patients fail acute treatment in the emergency department. These patients are usually admitted for a 3-5 day stay and receive extensive parenteral treatment. The goal of inpatient treatment is to control a disabling headache that has been unresponsive to other abortive therapy and is disabling to the child. Treatment protocols include the use of:

Prochlorperazine

Metoclopramide

Ketorolac

Valproate sodium ; injection.

Dihydroergotamine IV, Nasal spray.

SEVERE INTRACTABLE				
Prochlorperazine	0.15 mg/kg/IV; max dose 10 mg	Dopamine antagonist	Agitation, drowsiness, muscle stiffness, akinesia and akathisia	May have increased effectiveness when combined with ketorolac and fluid hydration
Metoclopramide	0.2 mg/kg IV; 10 mg max dose	Dopamine antagonist	Drowsiness, urticaria, agitation, akinesia and akathisia	Caution in asthma patients
Ketorolac	0.5 mg/kg IV; 15 mg max dose	Antiinflammatory and analgesic	GI upset, bleeding	
Valproate sodium injection	15 mg/kg IV: 1,000 mg max dose	↑ Brain GABA	Nausea, vomiting, somnolence, thrombocytopenia	Would avoid in hepatic disease
Dihydroergotamine IV	0.5 mg/dose every 8 hr (<40 kg) 1.0 mg/dose every 8 hr (>40 kg)		Nausea, vomiting, vascular constriction, phlebitis	Dose may need to be adjusted for side effects (decrease) or limited effectiveness (increase).
Nasal spray	0.5-1.0 mg/dose 0.5 mg/spray			

General life style advice

The importance of dietary triggers is generally over-estimated and probably only relevant during bad spell

Good sleep hygiene is helpful

Understand the different roles of prophylactic and acute medication

Sleep (if feasible in the context) will often cure a migraine episode

Adequate fluid intake avoid skipping meal, avoid triggers

Behavioral and cognition approach

Once a more chronic pattern has established, non pharmacological approaches are more likely to be effective

Identify the predisposing and precipitating factors through operation on the child and school environment

Unrecognized academic difficulties, difficulties with peers and home related stress are most common

Tension-type headache in children

Based upon criteria from the ICHD **bilateral, non-throbbing head pain of mild to moderate intensity, lasting 30 minutes to seven days**

TTH **is not** accompanied by **nausea or vomiting**.

Either photophobia or phonophobia may be present **(but not both).**

The headache **is not aggravated** by routine physical activity.

Prevalence

Prevalence in some studies shown as **high as 48**%

those having a combination of migraine and TTH around 20%.

Because of their mild to moderate nature, relative lack of associated symptoms and lower degree of associated disability **they are often ignored or have a minimal impact.**

Classification

Infrequent episodic (<1 headache day per month on average, **occurring <12 days per year**)

Frequent episodic (**1 to 14 headache days per month** on average, **occurring ≥12 and <180 days per year)**

Chronic (**≥15 headache days per month or ≥180 days per year)**

Diagnostic criteria

at least **10 episodes of headache**, **each lasting 30 minutes to seven days**, which fulfill the following conditions :

At least two of the following

Bilateral location

Pressing or tightening (non-pulsating) quality

Mild or moderate intensity

Not aggravated by routine physical activity such as walking or climbing stairs

•Both of the following:

No nausea or vomiting

No more than one of photophobia or phonophobia

These diagnostic criteria can be viewed as based more upon what TTH is **not**: localized, throbbing, severe, or aggravated by activity.

The physical and neurologic examination of a child with a history of TTH is **typically normal**. Some may have tightness and/or tenderness of the muscle at the base of the occiput or in the paracervical region

Neuroimaging — Routine neuroimaging studies **are not necessary** for the evaluation of a child with recurrent headaches when the neurologic exam is normal

Laboratory testing — Laboratory **testing is not necessary** in the evaluation of a child who meets criteria for TTH Likewise, electroencephalography (EEG) is not necessary or recommended for the routine evaluation of recurrent headaches, and there is no evidence to support performing a lumbar puncture **unless there is concern about increased intracranial pressure or infection.**

Treatment

simple analgesics (ibuprofen or acetaminophen) can be effective for acute treatment

Flupirtine is a nonopioid analgesic that has been approved in Europe

Amitriptyline has the most evidence of effective prevention of TTH

biobehavioral intervention, including biofeedback-assisted relaxation training and coping skills, can be useful as well.

Clinical features that may indicate intracranial pathology in children and adolescents with headache

Headache characteristics
Headache awakens the child or occurs consistently upon awakening from sleep
Short or paroxysmal headache; thunderclap headache (uncommon in children)
Associated neurologic signs and symptoms (eg, persistent nausea/vomiting, altered mental status, ataxia, etc)
Headache worsened in recumbent position or by cough, micturition, defecation, or physical activity
Absence of aura
Chronic progressive headache pattern
Change in quality, severity, frequency, or pattern of headache
Occipital headache
Recurrent localized headache
Lack of response to medical therapy
Headache duration of less than six months
Patient history
Inadequate history (description of headache and relative features)
Risk factor for intracranial pathology (eg, sickle cell disease, immune deficiency, malignancy or history of malignancy, coagulopathy, cardiac disease with right-to-left intracardiac shunt, head trauma, neurofibromatosis type 1, tuberous sclerosis complex, pre-existing hydrocephalus or shunt)
Age <6 years
Personality change
Deterioration of school work
Associated symptoms in the neck or back
Family history
Absence of family history of migraine
Examination findings
Child uncooperative (unable to complete neurologic examination)
Abnormal neurologic examination (eg, ataxia, weakness, diplopia, abnormal eye movements, other focal signs)
Papilledema or retinal hemorrhages
Growth abnormalities (increased head circumference, short stature or deceleration of linear growth, abnormal pubertal progression, obesity)
Nuchal rigidity
Signs of trauma
Cranial bruits
Skin lesions that suggest a neurocutaneous syndrome (neurofibromatosis, tuberosis sclerosis complex)

Evaluation

The first step in evaluating a child with headache is a thorough history and physical and neurological examination.

If these are abnormal or suspicious for a secondary cause, additional diagnostic testing is indicated.

The neurologic examination is the most sensitive indicator of needing further evaluation including neuroimaging.

Important Components of the History

- Age of onset:
  - Migraine frequently begin in the first decade of life
  - Chronic, non-progressive headaches begin in adolescence
- Mode of onset:
  - Abrupt onset may indicate intracranial hemorrhage
- Headache pattern

Acute

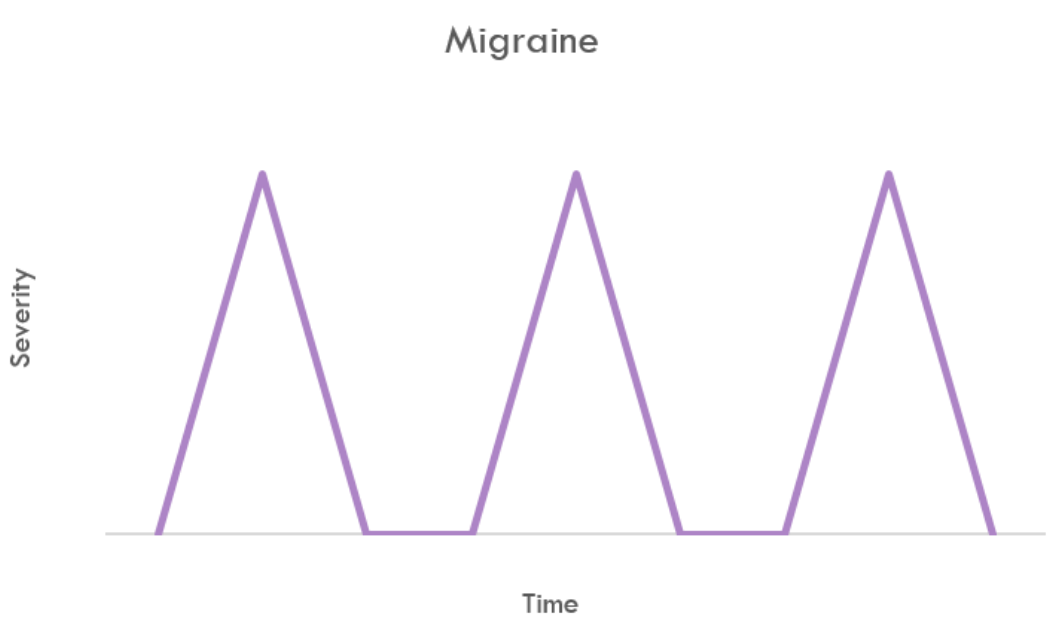
Acute recurrent (episodic)

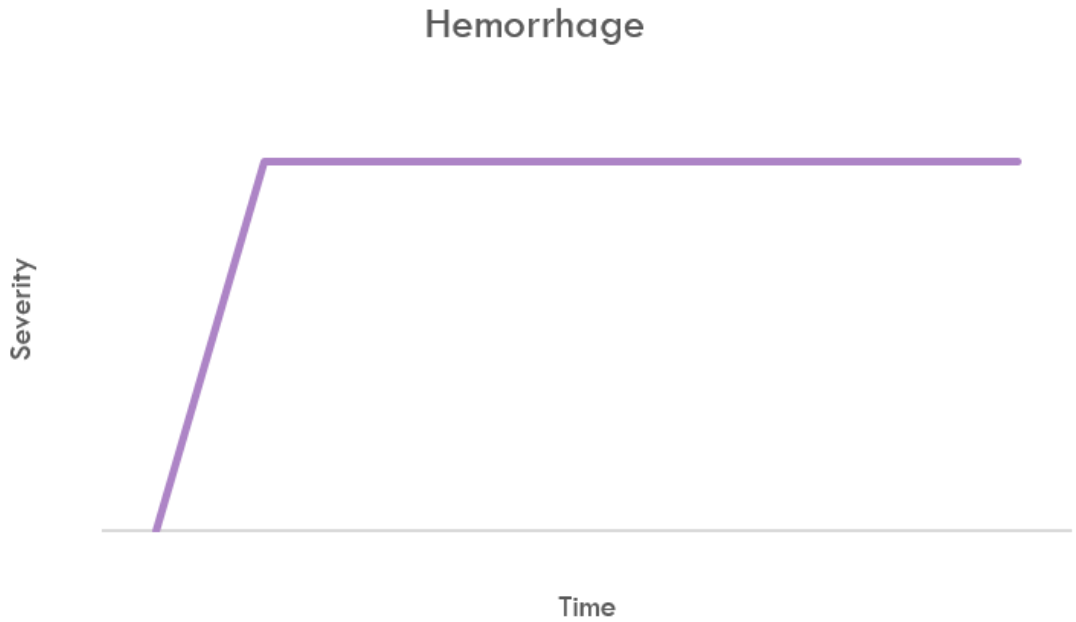
Chronic nonprogressive

Chronic progressive
  - Most primary headaches are episodic headaches that may become chronic
  - An acute change in an underlying recurrent, episodic headache disorder is cause for concern

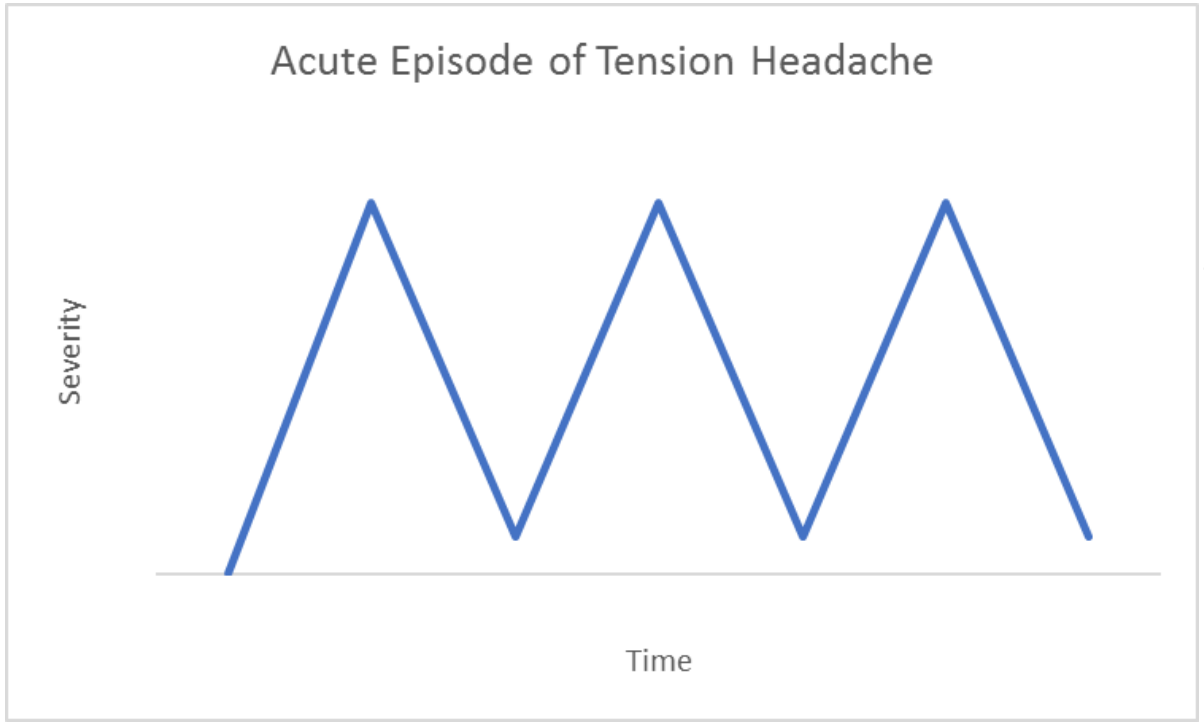
Important Components of the History cont.

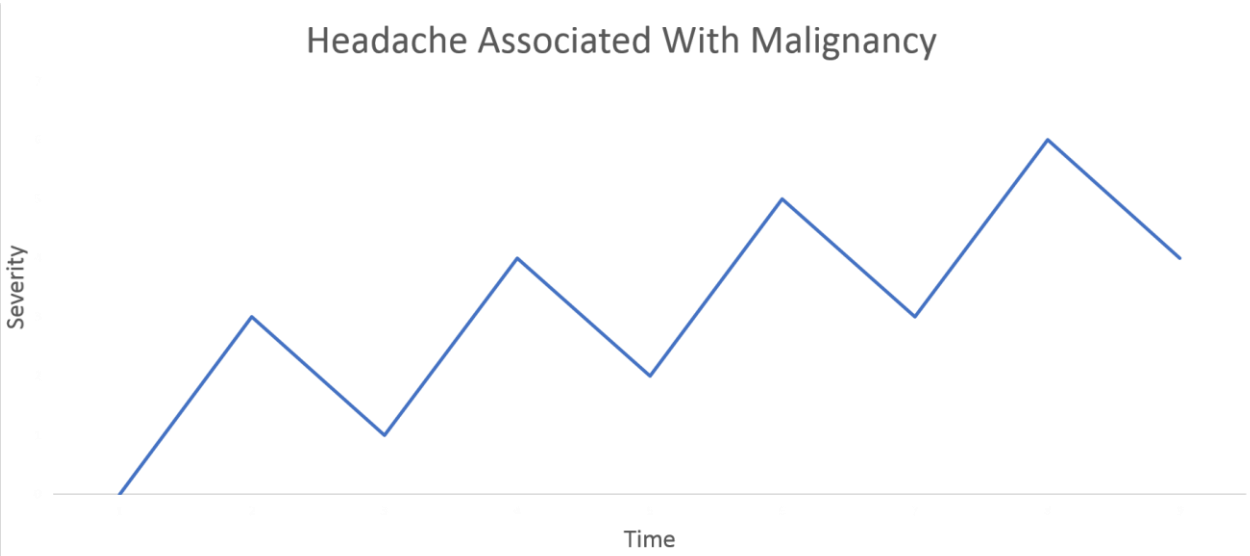
Severity & Course:





Important Components of the History cont.





Important Components of the History cont.

- Frequency:
  - Migraines occur 2-4 times a month, almost never daily
  - Chronic progressive headaches may occur 5-7 days a week
  - Cluster headaches occur 2-3 per day for several months
- Duration:
  - Migraines last 2-3 hours in young children and longer in adolescents
  - Tension headaches have variable duration
  - Cluster headaches often last 5-15 minutes but may last up to 60 minutes

Important Components of the History cont.

- Presence of aura/prodrome:
  - These are suggestive of migraine
  - If the symptoms are focal and repeatedly located on the same side of the body, a seizure or structural cause should be suspected
- Timing:
  - A headache that wakes a child from sleep or occurs upon waking may point to a space occupying lesion or increased intracranial pressure
  - Tension headaches usually occur late in the day

Important Components of the History cont.

- Quality:
  - Migraines have a throbbing/pulsating quality
  - Chronic nonprogressive headache have a tightness/squeezing pressures that waxes and wanes
  - Cluster headaches have a deep continuous pain
- Location:
  - Occipital: Posterior fossa neoplasm or basilar migraine
  - Orbital: Cluster headache
  - Localized: Secondary etiology eg sinusitis

Important Components of the History cont.

- Exacerbating factors:
  - Headaches with recumbent position, straining or valsalva indicate intracranial pathology
  - Migraines are usually triggered by certain foods, odors, bright lights, loud noises, sleep deprivation, menses or exercise.
  - Tension type headache worsens with stress, bright lights, noise and strenuous activity
  - Cluster headaches may worsen with lying down or resting
- Relieving factors:
  - Migraines usually respond to analgesic medication, dark & quiet room, cold compress or, sleep
  - Chronic nonprogressive headache responds to sleep but not analgesia.

Important Components of the History cont.

- Associated symptoms:
  - Neurological deficit may indicate increased ICP or a space-occupying lesion
  - Fever may indicate infection or intracranial hemorrhage (rarely)
  - Stiff neck may indicate meningitis, complicated pharyngitis or intracranial hemorrhage
  - Localized pain may indicate local infection such as otitis media, pharyngitis or dental abscess
  - Autonomic symptoms (such as nausea, vomiting, pallor, chills, dizziness, flushing or fever) may indicate migraine or cluster headache
  - Dizziness, numbness or, weakness may occur with idiopathic intracranial hypertension

Important Components of the History cont.

- Persistence of symptoms between headaches:
  - This is suggestive of increased ICP or a mass lesion
  - Resolution of symptoms between episodes is suggestive of migraine
- Impairment of normal function:
  - Chronic nonprogressive headache may cause frequent school absences
- Past medical history:
  - Certain underlying conditions are associated with increased likelihood of intracranial pathology (such as: sickle cell disease, immunodeficiency, malignancy, coagulopathy, head trauma, cardiac diseases with R->L intracardiac shunt, NF-1, tuberous sclerosis complex)

Important Components of the History cont.

Drug history:

- Drugs that may cause headache include:
  - OCPs
  - Steroids
  - SSRIs
  - SNRIs
- Drugs that may cause idiopathic intracranial hypertension include:
  - Growth hormone
  - Tetracyclins
  - Excessive Vitamin A
  - Steroid withdrawal

Important Components of the History cont.

Recent vision changes:

- May be associated with intracranial pathology like pituitary tumors, craniopharyngioma, or idiopathic intracranial hypertension.

Recent changes in sleep, exercise, or diet:

- These can be a trigger for headaches, and may point to a mood disorder

Changes in school or home environment:

- May be a source of stress

Important Components of the History cont.

Family history of headache or neurological disorders:

- Migraines, some tumors vascular malformations are heritable

What do the child and parent think is the cause of the pain:

- Indicated their level of anxiety about the headache

Mental health history and psychosocial stressors:

- Chronic nonprogressive headache may be associated with depression or anxiety

Important Components of the Physical Exam

General Appearance:

- Altered mental status may indicate meningitis, encephalitis, intracranial hemorrhage, elevated intracranial pressure, hypertensive encephalopathy.

Vitals:

- Hypertension may cause headache or be a response to increased intracranial pressure
- Fever suggests infection (most commonly upper respiratory infection) but may occur with intracranial hemorrhage or central nervous system malignancy

Important Components of the Physical Exam cont.

Visual fields:

- Visual field abnormalities may indicate increased intracranial pressure and/or a space-occupying lesion.

Fundoscopy:

- Papilledema may indicate increased intracranial pressure
- Funduscopic examination is normal in primary headache

Otoscopy:

- May demonstrate otitis media; hemotympanum may indicate trauma

Oropharynx:

- Signs of pharyngitis, dental decay or abscess

Important Components of the Physical Exam cont.

Neurological exam:

- Abnormal neurologic examination (particularly mental status, eye movements, papilledema, asymmetry, coordination disturbance, abnormal deep tendon reflexes) may indicate intracranial pathology

Skin exam:

- Signs of neurocutaneous disorders (eg, neurofibromatosis, tuberous sclerosis complex, which are associated with intracranial neoplasms)
- trauma (bruises, abrasions, etc).

Spine:

- Signs of occult spinal dysraphism (eg, midline vascular of pigment changes)

Neuroimaging

Neuroimaging studies may detect a variety of disorders that cause secondary headache. However, most children who present to primary care have primary or uncharacterized headaches and do not require neuroimaging

Decisions regarding neuroimaging in children with headaches should be made on a case-by-case basis

Urgent neuroimaging is reserved for patients with signs of increased intracranial pressure and/or focal neurologic examination with concern for a space-occupying lesion or intracranial hemorrhage.

Current Guidelines for Neuroimaging

Neuroimaging is indicated in:

- Children with abnormal neurological exam
- Children younger than 6 years of age
- Children who have worrisome features for an intracranial process
- Children with severe headache who have an underlying disorder that predisposes to intracranial pathology
- Child with occipital headache (debatable)
- Child with acute head trauma
- Child suspected to have an infection (complicated rhinosinusitis, encephalitis, some cases of bacterial meningitis)
- Chang in personality or behavior

Choice of Imaging Modality

Brain MRI is usually preferred.

Head CT is performed if MRI is not available or imaging is needed immediately.

MRI with gadolinium or CT with contrast should be performed if the clinician suspects an inflammatory cause or breakdown of the blood-brain barrier.

MRI is usually preferred in nonacute situations (or if there is persistent concern despite a normal head CT scan) because it minimizes radiation exposure and is more sensitive than CT. However, in young children, MRI may require sedation.

MR angiography or CT angiography may be indicated if subarachnoid blood or parenchymal blood is identified on initial MRI, CT, or lumbar puncture

Laboratory Evaluation

Laboratory testing rarely is helpful in the evaluation of childhood headache and is predominantly used to differentiate causes of secondary headache

Thank you